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THE

OPHTHALMIC REVIEW

A MONTHLY RECORD OF OPHTHALMIC SCIENCE

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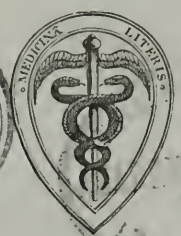

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CASES OF GLAUCOMA IN YOUNG PEOPLE.*

By John B. Story, M.B., F.R.C.S.I.
Surgeon to St. Mark's Ophthalmic Hospital, Dublin.

Primary glaucoma before middle age is of such rare occurrence that the following cases observed in individuals between the ages of 13 and 35 are worth recording. The first case is not one of primary glaucoma in the strictest sense of the term.

Case I.—*Acute glaucoma after subconjunctival injection of cocaine in a girl of* 13.

Miss Nina M., aged 13, has been under observation for some years. She suffers from convergent strabismus and hypermetropia. In May, 1890, the tendon of right internal rectus was divided under cocaine anæsthesia. A 3 per cent. solution of hydrochlorate of cocaine was used in the following manner. Three or four drops were first instilled into the conjunctival sac, and then somewhat less than 2 m. was injected subconjunctivally over the insertion of the internal rectus. Her pupils at the time were widely dilated by atropine used some hours previously.

About two hours after the operation I was sent for in haste to see her, and found her suffering from acute glaucoma, intense pain, and photophobia; several attacks of vomiting; T. + 1 or + 2, and pupil more dilated than at the operation. The pain was too great to permit of ophthalmoscopic examination. Frequent instillation of eserine made her all right in about two hours more, driving away the pain, reducing T. to normal, and contracting the pupil to about half its former size.

---

* Read before the Medical Section of the Royal Academy of Medicine in Ireland, December 16th, 1892.

About one month later I tenotomised the left internal rectus. This time no atropine was used at all, and 2 m. of eserine were instilled at the time of the operation to counteract the mydriatic action of the cocaine, which was used as on the former occasion. In spite of this precaution an attack of acute glaucoma came on some two hours after this operation also. Intense pain, and vomiting; T. + 1 or + 2; pupil dilated ad maximum towards internal side only, *i.e.*, the side next the situation of the subconjunctival injection of cocaine. It took 4m. of eserine every quarter of an hour to cure this attack of glaucoma, which lasted for one hour.

The horizontal diameter of cornea in each eye was 12 mm. and the refraction under atropine was (by retinoscopy) R., + 5 D. ⌒ + 0.5 cyl; L., + 4 D. ⊃ + 3 cyl.

CASE II.—*Chronic inflammatory* (?) *glaucoma in a woman of* 18.

Mrs. Hallinan, aged 18 (March, 1890). The history given by this patient was merely that she had been married one month, and that her sight failed subsequently, with pains in the eyes. She allowed on a subsequent examination that she had noticed blue rims to lamps so long ago as Christmas. The only account that her family could give of the disease was that it came on within one week of her marriage, and that she had desired to marry some man other than the bridegroom!

The case was a typical one of glaucoma. T. + 2 in both eyes; dilated and tortuous anterior ciliary vessels; pupils wide, the media clear, discs deeply excavated. Left pupil reacted slightly, the right not at all.

Vision: right eye = hand reflex in temporal field; left eye = fingers at 0.5m. in temporal field.

March 15th.—Large sclerotomy upwards in right eye, the iris being accidentally pricked by the point of knife; reduced T. to — 1, but left a large hyphæma.

March 16th.—Sclerotomy upwards in left eye. March 18th, T.n. in both.

March 20th.—Both pupils found more dilated than on 19th, in spite of eserine, and the hyphæma in right eye larger. T. approximately normal in each, tending to + in left and — in right. Vision unaltered by the operation.

March 24th.—Right T. — 2, hyphæma unchanged. No ophthalmoscopic illumination. Left disc deeply cupped with a few minute hæmorrhages under the temporal edge of disc spreading into retina. Marked atrophic ring round disc. By erect image, edge of disc — 12 ; bottom of cup — 20, but this examination was made while eye was well drugged with eserine. It took on all occasions a liberal use of eserine to produce any effect at all upon either pupil.

March 26th.—Hyphæma in right less, and disc visible, but the hyphæma was not completely absorbed in this eye even on March 31st, on which day we could see that a small prolapse of iris had occurred through the inner scleral incision.

The patient went home on April 2nd, with vision the same as before operation in each eye. Right T. + ? Left T.n. Refraction by erect image in right eye ; edge of disc — 3 ; bottom of cup — 8. In left eye, edge of disc — 4 ; bottom of cup — 8. Corneal diameter 12.5 mm. by Priestley Smith's keratometer.

Patient seen again Feb. 15th, 1892.

R. T.n. Small hernia iridis, due to sclerotomy. V. = hand reflex in temporal field. Priestley Smith's tonometer = 0.7mm. L., T. + ? V. fingers at 2m. Field very contracted as tested by fingers; nasally quite up to fixation point. Both pupils act. L. deciphers Wecker's type, No. 8; erect image; R., edge of disc — 5 ; bottom of cup — 8 ; L. edge of disc — 2 and — 3 ; bottom of cup — 6.

In contrast to these two extremely exceptional cases, I have made notes of two other cases of glaucoma, which are remarkable for the youth of the individuals affected —one a woman of 35, and the other a man of 30. This last case is also noteworthy, inasmuch as the patient's mother also suffers from the same disease.

Case III.—*Acute glaucoma in a woman of* 35.—Mary A. G., aged 35 (September, 1891). Complains of pain and much dimness of vision in her left eye for only two months. She had been treated (so far as I could make out, most injudiciously) by a distinguished surgeon in one of the Dublin hospitals. Her right eye was healthy. Left exhibited circumcorneal vascularity, dilated and tortuous anterior ciliary vessels, a hazy cornea, a dilated rigid pupil, a discoloured iris. V. = p. l., T. + 2. No details ophthalmoscopically visible; no effect produced on pupil or tension by a liberal use of eserine. Cornea obviously small, judging by the naked eye, but its diameter equalled 11.5mm. by Priestley Smith's keratometer.

Case IV.—*Chronic inflammatory glaucoma in a man of* 30. —Mr. B., aged 30, November 16th, 1892; has been wearing a convex cylinder, prescribed some five or six years ago by the late Mr. Redmond, for his left eye. This eye is weak and loses its sight every day towards evening. With correcting glass V. = 5/10? R. eye, V. = 5/7.5, and imperfectly = 5/5. His mother has noticed his left pupil getting big at the times when his sight is dim. He sees no coloured haloes. Slight chronic conjunctivitis on both sides, very shallow anterior chambers, and quite microphthalmic looking eyes; corneal diameter = 11mm. R. and L. T.n. R. and L., as also fundus. Venous pulsation in left disc. Ordered boric acid and nitrate of pilocarpine.

December 3rd.—He returned to say that he has an attack to-day—the second since using the pilocarpine, which has evidently done good, as the attacks were previously of daily occurrence. L. pupil wider than right. T. + 2. Tonometer measurement = 0.4mm. (while right = 0.6mm.) Left fundus exhibits both venous and arterial spontaneous pulsation—a phenomenon I do not remember to have ever seen before. The arteries—the larger ones—empty with a sudden jerk, then fill again immediately, and remain full till they empty, or rather diminish their diameter, with a sudden jerk again. I found it very difficult

to accurately compare the venous and arterial pulse. The vein only pulsated at one spot, just as it crossed one of the large arteries in the bottom of the disc, and from the arrangement of the vessels, and the impossibility of seeing down to the very bottom of the deep physiological (?) cup, it was impossible to be certain of the exact cause of the phenomenon. The portion of vein pulsating was posterior to the subjacent artery, *i.e.*, it was a portion of vein lying between that artery and the place where the vein passed out of the globe. It is hard to understand how any pulsation could be produced in that part of the vein by the action of that particular artery, and I would attribute the venous pulsation, not to the action of that artery, but of one lower down, nearer the exit of the vein, and ophthalmoscopically invisible, or to the nipping of the vein by some fibrous tissue as it passed through the lamina cribrosa. This much I was able to satisfy myself of—that the filling of the artery (and therefore of all the arteries) immediately preceded the filling of the vein. It looked as if the pulse wave in this artery, dilated the cavity of the vein. The vein only pulsated at this spot—deeper down than the spot where it touched the artery. The larger arteries pulsated both on the disc and for some distance also in the surrounding retina in the same jerky way described above. No pulsation visible in right retina.

December 4th.—After using eserine last night and this morning pupil contracted; T.n. (tonometer = 0.7mm.) and symptoms of coloured haloes round lights and dimness of sight quite disappeared. He had these haloes on this occasion. Pupil too small for thorough ophthalmoscopic examination, but so far as can be seen no pulsation present in retinal vessels.

An interesting point about this case is that the patient's mother has one eye blinded by glaucoma, and has chronic inflammatory glaucoma in the other one. I saw her during her second attack in this eye.

Since reading these notes, Mr. B. has been seen again, on February 1st, 1893, when he presented himself with an attack of a novel description. He had had no attack since

December 3rd, and had used pilocarpine drops daily till January 30th, when he forgot to put them in. That night he got an attack in his opinion exactly like the one of December 3rd, but it has continued ever since unabated, except in one respect, that the pain which marked its commencement has subsided. The eye (the left) looked just as on December 3rd: zonular vascularity; dilated pupil; V. = 5/30, disc slightly hazy, but cornea clear, with no keratitis punctata; very shallow anterior chamber, no visible pulsation, and, to my astonishment, T. — 1. Next day V. rose to 5/15, but T. remained — 1.*

The notes of the following case also have been added to since this paper was written.

Case V.—*Chronic Inflammatory Glaucoma in a man aged* 34, *associated with Keratitis Punctata.*—Mr. R. consulted me on December 3rd, 1892, complaining of fog before his right eye and a rainbow halo round lights. He had suffered from attacks of this description for some weeks, and was in all other respects a strong healthy man. R. eye V. = 5/15. T. + 1. Tonometer = 0.5mm. L. eye, V. = 5/7.5. T.n. Tonometer = 0.6mm. Corneal diameter = 12.5mm. right and left. Both pupils active, anterior chambers normal, discs deeply excavated. Right cornea hazy, but no keratitis punctata or iritic exudation visible, although most carefully searched for. Visible arterial pulsation in this disc, none in the other. Anterior ciliary vessels large in both eyes, and pupil of right slightly larger than that of left.

December 4th.—Right pupil contracted by eserine, vision with — 0.5 = 5/7.5—the same as that of left eye. T.n. in both. He says that the fog has been present off and on for more than a month, but that he believed it was caused by over indulgence in alcohol and tobacco.

December 7th. — Is worse to-day, and left eye also affected; sees coloured haloes before both eyes. Both corneæ

* Patient had a distinctly glaucomatous attack again on March 6th, with + T1, venous and arterial pulsation, dilated pupil and keratitis punctata. I advised him to submit to iridectomy.

hazy, but no keratitis punctata visible. T. + 1 in both. Right pupil contracted, so that pulsation of vessels of retina cannot be seen, but left disc exhibits spontaneous venous pulse and easily induced arterial pulse.

December 8th.—Both pupils contracted by eserine. V. = 5/5? in each eye. T.n. Tonometer = 0.6mm. and corneæ clear. This happy state of affairs continued unchanged for a week while using eserine, and for nearly a fortnight after discontinuing the myotic.

About Christmas time he got an attack in London, and consulted Mr. Tweedy, who kindly wrote to inform me that there was serous iritis in both eyes, and secondary glaucoma in the right. This attack passed off under the action of eserine, and he remained well till January 9th, when the right became again affected. A fresh attack occurred on January 13th, and on January 14th I performed a large iridectomy, using a Græfian knife on the right eye. Although the cornea was generally hazy during these attacks I could not detect any actual keratitis punctata or iritic exudation, therein failing to confirm Mr. Tweedy's observation. However, two days after the operation on right eye the left got a definite attack of serous iritis and keratitis punctata, which passed off rapidly without increase of tension under the action of atropine, and the right eye exhibited slight keratitis punctata five days after the operation.

Six days after the iridectomy he was walking about with dark glasses, the eye practically well and vision good except that accommodation was paralysed by atropine. An unfortunate accident occurred then. During his sleep he let his hand fall on his right eye, and awoke with intense pain to find the eye completely blind. When I saw him at 4 o'clock a.m., he could not even see the light of a candle. The wound had been burst open, and the anterior chamber was full of blood clot. T. + 1. For four days afterwards I could detect no diminution in the blood clot; it then began to absorb from above, and perception of light returned. After a week faint ophthalmoscopic illumination was perceived, and tension was normal. In a fortnight all

trace of blood had disappeared. T.n., media clear; V. = 5/50. After another week V. + 2 D cyl. = 5/10, T.n. During these three weeks the tension of left eye remained low and I kept it under the influence of atropine with most satisfactory results. At the end of the time no trace of keratitis punctata was visible, the pupil being widely dilated.

The cases recorded above are interesting in many and various respects. Case 1 is the first example I have met with of acute glaucoma produced by cocaine in any form, and is, so far as I know, the first case in which an attack has been caused by a subconjunctival injection of this drug. It is remarkable, too, because of the youth of the patient, and the absence of any peculiarity in the eyeballs which could account for the attack—I mean such an abnormality as microphthalmos, or unusually shallow anterior chambers. The little girl had been given a course of atropine drops to completely paralyse her accommodation on two former occasions without the slightest ill effects, and it is curious that the cocaine on this occasion should produce the effect which it did. It is interesting, too, to note the very local action of the cocaine on the iris of the second eye.

The second case of primary chronic inflammatory glaucoma in a woman of 18 is one of extreme rarity. Primary glaucoma is a disease of advanced life, and the following cases of various kinds of primary glaucoma are mentioned in Græfe-Sæmisch's "Handbook" by Schmidt as ophthalmological curiosities: (1) a girl of 9 seen by Stellwag; (2) a boy of 12 seen by Schirmer; (3) a boy of 16, and (4) a man of 19 seen by Mooren; (5) a woman of 20 seen by Peppmüller; and (6) a patient aged 22 seen by Schirmer. The youngest case seen by Priestley Smith ("Glaucoma," 1879) was a woman aged 31, and he refers (*loc. cit.*) to the case of a man of 25 reported by Critchett (R. L. Oph. Hosp. Rep., v. 224).

In Priestley Smith's paper on glaucoma in relation to

age, read before the Ophthalmological Society in 1886, and based on the records of 1000 unselected cases of primary glaucoma observed by himself and others, it appears that only five persons out of the 1000 were attacked by primary glaucoma under the age of 20. My patient was most probably attacked by the disease long before she came to consult me. I believe she had suffered for a considerable time from simple chronic glaucoma, and the more acute form she finally developed only appeared about the time immediately preceding or following her unfortunate marriage, when we may assume that congestive phenomena would be very likely to show themselves in the ocular regions.

Case III. presents no features of interest beyond the youth of the patient, aged 35, and it is not impossible that the glaucoma may be secondary to the growth of an intraocular tumour. She would not come into hospital, and has been since lost sight of.

Case IV. is interesting from the youth of the patient, aged 30, the hereditary nature of the disease, and the association of both venous and arterial visible pulsation. The arterial pulsation was not confined to the disc, but was visible in all the larger arteries to a considerable distance from the disc, thereby refuting what is stated in Græfe-Sæmisch (" Handbook," vol. vi., p. 8), that the glaucomatous arterial pulse is only visible in the disc.

This case is also interesting from the support it lends to the views of Priestley Smith upon the etiology of glaucoma. Mr. B.'s eyes were obviously microphthalmic, *i.e.*, they appeared microphthalmic to ordinary inspection, and the corneal diameter was in each of them below that of the average eye.

As is well known, Mr. Smith brought forward the statistics which have been quoted above in support of a theory that glaucoma depends to a certain extent upon the presence of a large crystalline lens, and a narrowing of the circumlental space. He has fully established two

points: (1) that glaucoma is pre-eminently a disease of advanced life, and that to an extent not formerly realised by oculists; (2) that the lens steadily increases in volume and diameter up to extreme old age, while it is known that the connective tissue structures surrounding it attain their full dimension early in adult life.

Mr. Smith regards a narrowing of the circumlental space as the underlying cause of glaucoma, and the determining conditions may be either an abnormally large lens, an abnormally small ciliary region, or an abnormal enlargement of the ciliary processes.

It is unlikely that an abnormally large lens was present in any of the cases here recorded. The age of the patients renders such a supposition improbable, and the only case which shows any evidence of an abnormally small ciliary region is Case IV. The only indication we have of the size of the ciliary region in the living eye is the diameter of the cornea. The average diameter of the cornea, as found in 1,000 eyes normal, except for refractive errors, is 11.6mm. (*vide* Priestley Smith, *loc. cit.*). The diameter in Case IV. was only 11mm. The diameter in all the others, except the doubtful Case III., was above the average.

The spontaneous occurrence of the low tension in Case IV. is a phenomenon for which I can advance no explanation. The state of the eye as regards shallow anterior chamber and dilated pupil was just the same as during the previous attacks of undoubted glaucoma. The future of this eye will be a matter of considerable interest, and I hope to have an opportunity of reporting upon it again.

Case V., too, offers matter for future investigation, both as to the fate of the eye operated on (whether cataract may not form in it as the result of the blow during sleep), and as to the condition of the second eye, which has had both high tension and keratitis punctata, and may develop either or both affections again. The

occurrence of high tension as a sort of forerunner of keratitis punctata may have some bearing upon Smith's theory of a narrow circumlental space, and is a novelty in my experience.

A table is appended of the size of the corneal diameter in all the glaucoma cases where I have measured it. The number is not large, but the figures are given in the hope that other oculists of greater experience may be induced to let us know their observations upon this point.

Thirty-one eyes affected by glaucoma were measured, and the average corneal diameter amounts to the figure of 11.9mm., or 0.3mm. above the average diameter as ascertained by Priestley Smith. The largest diameter measured was 13mm., and this only occurred once. The smallest was 11mm., and this was found in eight eyes.

No. of Eyes.		Age.		Eye.	Corneal Diameter in mm.
1	Ellen S., D.C., 3,340, Oct. 1891	60	Acute Glaucoma	R.	12.5.
2	Do.		Do.	L.	12.5.
3	Bessie H., D.C., 3,841, Oct. 1891	50	Acute Glaucoma	R.	12.
4	Do.		Do.	L.	11.5.
5	Michael C., D.C., 3,963, Oct. 1891	71	Chronic Glaucoma	R.	12.
6	Do.		Do.	L.	12.
7	Mary G., D.C., 2,715, Sept. 1891	35	Acute Glaucoma	L.	11.5.
8	Anne K., H.C., 379, Oct. 1891	60	Acute Glaucoma	R.	11.
9	Do.		Do.	L.	11.
10	Pat. L., H.C., 400, Oct. 1891		Chronic Glaucoma	R.	12.
11	Do.		Do.	L.	12.5.
12	Pat. C., H.C., 426, Nov. 1891.	75	Chronic Glaucoma	R.	12.5.
13	Mary B., H.C., 464, Dec. 1891.	77	Chronic Glaucoma	R.	12.5.
14	Do.		Do.	L.	12.5.
15	Elizabeth K., D.C., 3,043, Nov. 1891.	62	Chronic Glaucoma	R.	12.5.
16	Do.		Do.	L.	12.
17	Mrs. J., Sept., 1892 (A.H.B.)	72	Chronic Glaucoma	R.	12.
18	Mrs. D., Sept., 1892 (A.H.B.)	60	Acute Glaucoma	R.	11.
19	Do.		Do.	L.	11.
20	Mr. R., Case V.	34	Chronic Inflam. Glau. & Keratitis Punctata	R.	12.5.
21	Do.			L.	12.5.
22	Mrs. B., Dec. 1892	50	Chronic Inflam. Glauc.	R.	11.
23	Do.		Do.	L.	11.
24	Mr. M., Nov., 1892.	64	Chronic Glaucoma	R.	12.
25	Do.		Do.	L.	13.
26	Mr. B., Case IV.	30	Chronic Inflam. Glauc.	L.	11.
27	Mrs. B., Mother of Case IV.	62	Chronic Inflam. Glauc.	L.	12.
28	Mrs. H., Case II.	18	Chronic Inflam. Glauc.	R.	12.5.
29	Do.		Do.	L.	12.5.
30	Nina M., Case I.	13	Acute Glaucoma after Cocaine	R.	12.
31	Do.			L.	12.

CONGENITAL PTOSIS WITH ASSOCIATED MOVEMENTS OF THE AFFECTED LID.

By Walter N. Sinclair, M.B.
Senior House Surgeon Birmingham and Midland Eye Hospital.

The following cases may be of interest as illustrating a somewhat remarkable condition.

Case I.—M. A., aged 7, a delicate, strumous little girl, came complaining of drooping of the left upper lid and uncontrollable movement thereof when eating. There is well-marked left ptosis (present at birth), and the child has no voluntary power of raising the eyelid. On giving her a biscuit to bite, it is seen that with every movement of the lower jaw downwards and to the right the eyelid is forcibly raised. Some idea of the extent of this movement can be gained from the fact that while the eyelid covers quite a third of the cornea when the eyes are directed forwards, with every movement of the jaw it rises so as to reveal two mm. of sclerotic above the cornea. The movement is still more evident if the child looks down. There is no synchronous contraction of the pupil, which is equal to its fellow in diameter. The patient has no control over the movement. She has hypermetropic astigmatism, slightly greater in amount in the left eye.

R. V (corrected) = $\frac{6}{8}$
L. V (,,) = $\frac{6}{12}$

Fundi normal.

Case II.—J. F., aged 34, a strong healthy man, came on account of visual defect in the left eye.

R. V = $\frac{6}{6}$; no Hm.
L. V = $\frac{6}{8}$

after hypermetropic astigmatism has been corrected. Fundi normal.

The left eye shows slight ptosis, dating from birth. He has no voluntary power of raising the eyelid in any position of the head, nor when the fellow eye is covered.

With every movement of the jaw downwards the drooping lid rises, this being more marked when the eyes are directed downwards. The ptosis is so slight in this case that when the patient looks up no distinct movement can be detected when the jaw is depressed, though a slight contraction of the levator can be felt. The eyelid is raised nearly as much when the jaw is moved from side to side while the mouth is kept shut, each movement to the right (left external pterygoid) causing the lid to rise. The patient is quite conscious of the movement but has no control over it. No consensual contraction of the pupil with the movement.

Case III.—A. B., aged 22, a healthy young man, marked left ptosis, present from birth. Patient cannot voluntarily raise the drooping lid in any position of the head, whether the fellow eye be covered or not. Very distinct movement of the eyelid upwards when the lower jaw is depressed, or when it is moved to the right side (left external pterygoid), digastric. No synchronous contraction of the pupil. In addition to the involuntary movement of the left upper lid, this patient has a nervous habit of forcibly squeezing up his right eye once or twice a minute. So far as could be ascertained, no test type being available, vision was normal in both eyes.

Some fifteen cases only of this curious condition are on record (Swanzy, 1892), but the fact that the three cases came to my notice within six months makes it probable, as Nieden has already suggested, that such cases are less rare than is commonly supposed. It will be noted that all three cases agree in the following particulars: (1) There is congenital ptosis. (2) The *left* upper lid is affected. (3) While the eyelid cannot be voluntarily raised, it moves upwards freely when certain of the masticatory muscles are thrown into action.

As regards the first point, in only two of the recorded cases of associated movements, so far as I have been able to ascertain, was ptosis absent: (1) Fuchs; (2) Fränkel.

With regard to the second point, in a large majority of the cases recorded the left eyelid has been the affected one. At least one case, however, is on record in which the right levator was the abnormal one (Ole Bull, *Archives of Ophthalmology*, vol. xvii.), and it is difficult to explain why the left side should have a monopoly of this peculiarity.

As to the last point, in most of the cases described it is not stated whether the eyelid could be voluntarily raised when the fellow eye was covered. Ole Bull has described two cases very similar to mine, but in which voluntary power over the levator was present when the other eye was covered. From this fact he argues that the condition is a purely reflex one—a view shared in by Rosenmeyer and Nieden, and made plausible by the frequent association of movement of the eyelids and jaws observed in children and patients suffering from blepharospasm, &c. In two of my cases (the third was unfortunately not tested as regards this point) no such voluntary power was present. This, together with the almost invariable presence of ptosis, seems to point to an unusual distribution of nerve fibres as the explanation of the condition. This is the view held by Gowers and Helfreich, and supported by the Committee of the Ophthalmological Society in their report on a similar case recorded by Mr. Gunn (*Transactions*, vol. iii.). The explanation given by them is that the levator is wholly, or in part, innervated by fibres from the fifth nerve, and this hypothesis is strengthened by the numerous and intimate connections which exist between the two nerves and the ganglia at their entrance into the orbit. Still in many respects the condition is a most remarkable one, and any information as to associated movements of other muscles would be valuable.

For permission to record the above cases I have to thank Mr. Lloyd Owen.

O. SCHIRMER (Halle). A Clinical and Pathological Study of the Pathogenesis of Sympathetic Eye Inflammations. *von Graefe's Archiv.*, *vol. xxxviii.*, *part* 4, *p.* 93, 1892.

Acting on the suggestion of Professor Von Hippel, and with the help of the resources of the Königsberg Eye-clinic, Schirmer has examined afresh the whole of the evidence relating to the nature of sympathetic ophthalmia. He has made a critical analysis of the literature of the subject, and has supplemented this by the collation of many cases hitherto unpublished, and by certain original observations and experiments. The result is a comprehensive and important article covering more than 200 pages. We shall attempt to reproduce here the chief points in the argument and the conclusions.

The re-investigation of the whole subject is justified by the fact that whereas a few years ago Deutschmann's theory was pretty generally received as satisfactory, the old diversity of opinion appears now again to prevail. The artificial induction of sympathetic disease in animals by Deutschmann's method has failed with nearly all those who have attempted it. The search for micro-organisms in eyes which have excited sympathetic inflammation has in many cases given negative results, and the bacterial nature of the disease is now again disputed by many observers.

Sympathetic inflammation is clinically to be sharply distinguished from sympathetic irritation. The latter is a variable condition, but has in all its forms two well-marked characters. It presents no true inflammatory changes, and it is entirely dependent upon the continued influence of the exciting eye, being quickly and completely cut short by the removal of this latter. In analysing published cases of so-called sympathetic disease, this distinction must be constantly borne in mind, or wrong conclusions are likely to be drawn. In establishing the diagnosis of sympathetic inflammation three points demand especial attention, viz., the nature of the affection

of the exciting eye, the nature of the affection in the sympathising eye, and the interval of time between the affection of the two eyes respectively.

In order to avoid confusion it must be noted here that Schirmer's use of the verb "to sympathise" is peculiar. According to him, the eye which excites sympathetic mischief is "the sympathising eye" (*das sympathisirende Auge*), while the eye secondarily affected is "the sympathised eye" (*das sympathisirte Auge*). In English we speak of the second eye as *sympathising with*, and not as being *sympathised by*, its fellow. We shall, therefore, translate Schirmer's terms by the words "exciting" and "sympathising" in their ordinary sense.

The Disease in the Exciting Eye.

Seeing that the question of first importance is that of the infective or non-infective nature of the disease, it is necessary to divide the cases into two groups: (1) Cases in which the tunics of the eye have not been perforated; (2) Cases in which the tunics have been perforated. The first group includes the following disorders, all of which have been known, or supposed, to cause sympathetic inflammation.

Herpes Zoster Ophthalmicus.—Of four recorded cases, one presented only sympathetic irritation; the second gave no definite evidence that the second eye was influenced by the first; the third presented nothing more than a phlyctenular inflammation in the second eye; and in the fourth there had been perforation of the cornea in the exciting eye, followed by shrinking of the globe. There is therefore no evidence that herpes, as such, can cause sympathetic inflammation.

Symblepharon.—Here, also, there is no satisfactory evidence of the occurrence of sympathetic inflammation, apart from those cases in which the burn or other severe injury which leads to the symblepharon, causes also a perforation of the globe.

Choroidal Sarcoma. Retinal Glioma.—A careful search brings to light twenty-eight cases attributed to sarcoma, two to glioma. The analysis of these shows that nine were cases of sympathetic irritation; that some others were of doubtful character; and that in several more a perforation of the exciting eye had occurred either spontaneously or by operation. Excluding these, there remain at least three cases in which a choroidal sarcoma, with no perforation of the tunics, appeared to cause a true sympathetic inflammation in the fellow eye. In these three cases the exciting eye presented, in addition to the tumour, a well marked irido-cyclitis, and this complication was present also in some of the more doubtful cases just referred to. It appears, therefore, that a choroidal sarcoma can only cause sympathetic inflammation when it is associated with irido-cyclitis, and seeing that only a small minority of such tumours are complicated in this way, the irido-cyclitis and not the tumour must be regarded as the chief agent. What, then, is the connecting link between the tumour and the irido-cyclitis? It is difficult to believe that a sarcoma can of itself excite inflammation in the uveal tract, for such inflammation is a very exceptional complication. We may suppose that the uveal tract suffers endogenous infection through the presence of organisms in the system at large, and that the presence of a tumour provides specially favourable conditions for such infection. A migratory infection from the one eye to the other is then a reasonable supposition. On the other hand, if the one eye is infected from the system, we cannot exclude the possibility that the fellow eye is also directly infected in the same way. The nature of this exceptional condition must therefore remain doubtful.

Intraocular Cysticercus is sometimes mentioned as a cause of sympathetic ophthalmia, but it appears that only one case of the kind has been recorded, and that in this there was merely some sympathetic irritation.

Subconjunctival Rupture of the Globe.—An analysis of the published cases shows that in some there was merely a

sympathetic irritation; that in others the affection of the second eye was perhaps not sympathetic at all; and that in others, again, the conjunctiva had been perforated, either at the time of the injury, or later, for the extraction of the lens. In the majority of cases of subconjunctival rupture, healing takes place without inflammatory reaction. In the exceptional cases which have caused mischief in the fellow eye, the injury has always been followed by irido-cyclitis in the exciting eye. Evidently it is not the injury *per se*, but the irido-cyclitis, as in the case of tumour, which threatens the fellow eye. The question of excising an eye injured in this way should therefore be decided by the presence or absence of this inflammatory complication.

Bone Formation.—The usual history in such cases is that the eye was injured years ago, and that irido-cyclitis and ultimately shrinking followed. That slight inflammatory attacks recurred at intervals, or that after a long period of quiescence inflammation began again with injection, watering, and photophobia, and that some weeks or months later the fellow eye became affected. The exciting eye is always irritable, and generally tender on pressure at the time when it affects the fellow eye. In the absence of such irritability, the mere presence of a bone formation does not suffice to cause sympathetic mischief. The propriety of excising such a shrunken eye should therefore be determined, as in the previous case, by the presence or absence of injection and tenderness.

Spontaneous Inflammations.—A spontaneous inflammation in one eye is sometimes supposed to be the cause of a sympathetic inflammation in the other. It would be unwise to deny the possibility, but on the other hand it seems impossible to prove the connection, for in every such case the inflammation of the second eye may with at least as much reason be assigned to the same cause as that of the first. Sympathetic irritation may no doubt arise from a severe spontaneous inflammation in the fellow eye.

The second group of cases, those namely, in which the exciting eye has been perforated, must now be considered.

Operations.—An operated eye which has excited sympathetic inflammation in its fellow presents one constant characteristic—the presence of inflammatory changes. In a large number of cases, a plastic irido-cyclitis has quickly followed the operation and spoiled the eye; in other cases the immediate result of the operation has been good, but weeks or months later an insidious uveitis has set in. In the very few exceptional cases in which, without any inflammatory change in the operated eye, the fellow eye has become inflamed weeks or months after the operation, it is reasonable to doubt the sympathetic nature of the affection, for an operation upon one eye does not protect the fellow eye from spontaneous inflammation, and such a sequence will in the nature of things sometimes occur. It may, then be, asserted that the link between an operation and a sympathetic inflammation is a uveitis in the operated eye. It is now generally admitted that every severe uveitis which immediately follows an operation is of infective nature. With regard to the more insidious form which sometimes attacks an operated eye at a later period there may be some question, but there is strong reason for the belief that the usual cause is a late infection occurring at the cicatrix, where a slight prominence or want of solid closure, supplemented by a temporary excoriation of the epithelial covering, may at any time afford a ready entrance for infective organisms.

Traumatic Plastic Uveitis.—It is well established that it is the inflammation which follows a perforating wound, and not the wound itself, which threatens the fellow eye, and that such inflammation is usually due to infection through the wound. The danger attaching to the presence of a foreign body in the interior of the eye has probably been somewhat overrated, for there is no evidence that aseptic foreign bodies can cause sympathetic inflammation, even though, as in the case of fragments of copper, they may by chemical action damage the injured eye.

The danger lies in the fact that very many foreign bodies carry infective organisms with them into the eye. The only cases in which a foreign body was supposed to affect the fellow eye without the intervention of uveitis in the injured eye, were cases of sympathetic irritation only. The most dangerous cases are those in which the injured eye seems at first to tend towards recovery, but nevertheless remains more or less injected and tender on pressure. This tenderness is the expression of a persistent cyclitis, and is a most important indication. Its absence cannot, unfortunately, be taken as a proof that there is no cyclitis, for a slight cyclitis does not always reveal itself by tenderness on pressure.

Traumatic Purulent Uveitis.—This is the most severe form of infective inflammation, yet it is a less frequent cause of sympathetic inflammation than the foregoing. In order to reconcile these facts with the migratory theory of sympathetic ophthalmia, Deutschmann supposes that the infective organisms are annihilated or extruded by the very intensity of the inflammation, the free formation of pus, and the rupture of the tunics. Gifford supposes that the channels of communication, the lymph spaces, become blocked by the inflammatory products. But the evidence in this direction is not satisfactory, for the staphylococci have been found still active after a panophthalmitis of four weeks' duration, and they have been found capable of making their way into the sheath of the optic nerve. Sattler holds that sympathetic inflammation is caused by a specific organism. If this be so, a sympathetic ophthalmia following a suppurative uveitis must be attributed to a mixed infection of the exciting eye, and the absence of sympathetic ophthalmia in such a case may be due either to the absence of the specific germ in the exciting eye, or to its destruction by the staphylococcus.

Gonorrhœal conjunctivitis leads to corneal perforation, and hence to infective irido-cyclitis. Sympathetic inflammation may follow. There is, however, no evidence that the gonococcus itself invades the other eye ; indeed, the evi-

dence seems opposed to this idea, for this organism, while it excites an intense suppurative inflammation, has little tendency to penetrate deeply into the tissues. The perforation of the cornea exposes the eye, as in all other cases, to infection by various organisms, and the occurrence of sympathetic mischief in connection with gonorrhœal conjunctivitis is probably an example of mixed infection.

Tuberculosis has been supposed to excite sympathetic inflammation. In the two recorded cases, however, the tunics of the exciting eye were perforated, and the inflammation in the fellow eye was of the usual sympathetic type, and presented nothing characteristic of tuberculosis.

Leprosy has been mentioned in the same connection, but apparently by mistake; the evidence is entirely negative.

Anophthalmos.—The wearing of an artificial eye over a shrunken globe sometimes appears to excite sympathetic inflammation. Such stumps are liable under any circumstances to recurrent inflammation, and may probably sometimes undergo late infection through the damage inflicted by a roughened artificial eye. These cases, however, should not be classed with anophthalmia. There is no satisfactory evidence that true sympathetic inflammation can be excited from an anophthalmic orbit.

The foregoing facts confirm the assertion of von Graefe, that an inflammation in the uveal tract of the exciting eye is a necessary factor in the production of sympathetic inflammation. They also show it to be highly probable that this uveitis depends on microbic infection, and not upon mechanical or chemical irritation. These conclusions, however, give, so far, no information as to the manner in which the inflammation is transmitted to the second eye.

The Interval between the Affection of the First Eye and of the Second.

A considerable number of trustworthy records show an interval of three weeks between the affection of the ex-

citing eye and that of the sympathising eye; in a few cases the interval was less than this by two or three days; two recorded cases show an interval of fourteen days, but in these latter the evidence is not quite conclusive.

The maximum interval is more difficult to determine. In many recorded cases the presence of a shrunken globe, devoid of all signs of irritation, has been held to justify the diagnosis of sympathetic inflammation in the fellow eye; but such a connection can hardly be admitted. On the other hand it is certain that shrunken globes are liable to recurrent attacks of inflammation after long periods of time, and that sympathetic inflammation may be induced thereby. The recurrence in the exciting eye may probably be due to a reinfection from without, in the manner already referred to; or it may be due to germs which have remained in the eye, and have multiplied with lapse of time sufficiently to excite fresh inflammation. Living germs have been found in many eyes excised long after the receipt of the injury.

Preventive Enucleation.—The outbreak of sympathetic inflammation after the removal of the exciting eye has been many times recorded. It may occur on the day following the enucleation, or at any time during the first three weeks. In a few cases the interval has been some days longer still. In two instances of special interest the eye was removed within a day or two after a severe injury, which caused also a severe and somewhat persistent orbital cellulitis; sympathetic inflammation began at the end of five and seven weeks respectively, and was attributed to a transmission of the infective inflammation of the orbital tissue. It has been asserted that unless sympathetic irritation is present before the enucleation, sympathetic inflammation cannot occur afterwards, but there are several cases on record which disprove the statement. It is of course true that a sympathetic inflammation which is mild before the enucleation may become severe at a later period.

Preventive Evisceration.—Two cases of sympathetic inflam-

mation after evisceration are on record, but seeing that the outbreak occurred in each case within the period in which it may occur after enucleation, they do not prove that evisceration affords less safety than the entire removal of the eye, though on theoretical grounds some difference in this respect may be assumed.

Preventive Neurotomy and Neurectomy.—An optico-ciliary neurotomy has been followed in a considerable number of cases by sympathetic ophthalmia, and it would seem that the operation itself, rather than the condition of the eye thus treated, was in some cases at least to blame. It is well established that reunion between the divided ends both of the optic and of the ciliary nerves is apt to take place. Even a neurectomy affords no absolute security, for in one case in which the optic nerve was removed to the extent of 15mm., sympathetic inflammation began four weeks after the operation. But as it is very probable that this operation diminishes the risk to the fellow eye, it may justifiably be performed as a substitute for enucleation when the patient declines to submit to the latter.

The Disease of the Sympathising Eye.

The fundamental difference between sympathetic irritation and sympathetic inflammation must here again be insisted on. The latter condition may be induced by various affections in the fellow eye of different nature from those which induce the former, *e.g.*, by the presence of a foreign body beneath the lid. It may co-exist with sympathetic inflammation; it may exist for a long time without the occurrence of the latter; it may precede it by a shorter or longer time; it may be completely absent before and during the occurrence of sympathetic inflammation.

Sympathetic inflammation usually presents itself as a uveitis or a papillo-retinitis, or as both combined. Before these typical conditions are discussed, the following alleged varieties must be mentioned.

Sympathetic Conjunctivitis.—In the published cases the diagnosis seems to have been based chiefly on the fact that

the conjunctivitis improved after enucleation of the other eye—at best a very weak argument. A direct transference of secretion from one eye to the other, or the rubbing and spasm of the lids which accompany the irritation of a shrunken globe would seem a more reasonable explanation of a conjunctivitis in the fellow eye.

Sympathetic Keratitis.—There is no proof of any direct connection between the affection in the first eye and the supposed sympathetic ulceration, infiltration, &c., in the fellow eye. The enucleation of the first would remove any sympathetic irritation which might co-exist with these corneal changes, and the admission into hospital would be likely to directly benefit them.

Sympathetic Cataract has no proved existence apart from the secondary cataracts which are due to sympathetic uveitis.

Sympathetic Retinal Detachment, in so far as it exists, is in like manner a complication and consequence of sympathetic uveitis.

Sympathetic Glaucoma.—In an eye predisposed to glaucoma almost any nerve disturbance may induce an acute attack, and amongst other causes an irritation originating in the other eye may no doubt be efficient. A secondary glaucoma may occur as a consequence of sympathetic iritis, with exclusion of the pupil.

Sympathetic Simple Atrophy of the Optic Nerve has been supposed to occur, but the evidence is quite insufficient.

We come now to the changes in the sympathising eye, about the existence of which there is no question.

Sympathetic papillo-retinitis is not unfrequently to be found co-existing with sympathetic uveitis, and may probably, in some cases at least, be regarded as a product of the latter; for we often see a papillitis of similar type in cases of spontaneous irido-cyclitis. Possibly the same causation may be at work in some of the cases in which

no uveitis is discoverable, for it is certain that in some cases of choroiditis no changes, apart from those in the retina, may be at first discoverable. Yet there seems to be ample evidence that there is such a condition as a primary sympathetic papillo-retinitis. It has certain peculiar characteristics. It has never yet been known to begin after the removal of the exciting eye. It is a benign affection, being cured by the enucleation. It is not immediately cured, like sympathetic irritation, but disappears gradually in the course of some days, for it is a condition of actual inflammation. These characters strongly suggest that it depends upon the transmission from the exciting eye, not of bacteria, but of bacterial products. Experiments have shown that fluid injected into the subpial space of one optic nerve can travel round by the commissure and other optic nerve to the papilla of the fellow eye without penetrating far along the optic tracts or into the meningeal spaces. Other experiments have shown that certain irritant fluids injected into the one eye may travel round along this same route and set up papillitis in the fellow eye. The above-mentioned explanation of sympathetic papillitis is in complete accord with these experimental results. The slight intensity of this form of papillitis, as compared with that arising from brain disease, as also its speedy disappearance after the removal of the exciting eye, may reasonably be attributed to the slight concentration of the irritant—the phlogosin derived from organisms located in the fellow eye.

Sympathetic Uveitis.—Three forms of uveitis—serous iritis, plastic iritis, and plastic or malignant irido-cyclitis—are commonly described, but no hard line can be drawn between them, for each form is liable to pass on to another. A malignant irido-cyclitis may begin as a serous iritis, and what appears clinically to be a serous iritis, or more properly, according to some writers, a serous cyclitis, may prove an anatomical examination to be complicated by choroidal changes. A suppurative form of sympathetic uveitis has been described, but it is probable that the

supposed hypopyon was only such a deposit of thick secretion as may occur in severe plastic iritis. Moreover, the course of the supposed suppurative disease was chronic in all cases, and was, in one case, cured by mercury.

Sympathetic Uveitis usually involves all three parts of the uveal tract. It commonly appears to pass backwards from the iris and ciliary body to the choroid, but may begin further back and extend forwards. In some cases it appears to be limited to the iris; in others to affect the choroid and to involve the retina and papilla without invading the anterior parts of the uveal tract. It is usually a malignant affection running a long course, and, even when appearing to subside, having a great tendency to relapse. It is probable that some of the cases recorded as cured have subsequently relapsed and ended disastrously. There are, however, many instances in which a malignant plastic uveitis has undergone permanent cure. The serous form is comparatively benign, but may pass into the plastic form.

The supposed migration of living infective organisms from one eye to the other, by way of the optic commissure, would appear to involve the likelihood of cerebral affection, but, whatever be the reason, the fact is that apart from headache, a feeling of pressure, and occasionally vomiting—symptoms which arise in the course of many severe intraocular disorders without any trace of meningitis—cerebral symptoms have hardly ever been noted in connection with sympathetic ophthalmia.

Conclusions concerning the Nature of Sympathetic Inflammation.

The various theories of sympathetic inflammation attribute the disease to one or other of two fundamentally different agents—nerve irritation and bacterial migration.

Opposed to the nerve theory are the following facts. Irido-cyclitis in the exciting eye is an essential condition, and in the vast majority of cases this irido-cyclitis is cer-

tainly of bacterial origin. Mechanical disturbance of the ciliary nerves, even though associated with intense pain, as in glaucoma, does not induce the disease. If, therefore, the ciliary nerve theory is to be upheld, we must assume that the peculiar nerve influence which induces the disease can be set in operation only by bacterial irritation in the exciting eye, and this assumption can be supported by no analogy. A transmission of any such nerve influence along the optic nerve is negatived by the fact that in many cases this nerve is completely atrophied before the outbreak of the disease. Again, the disease is characterised by a great persistency, and a great tendency to relapse even after the removal of the exciting eye, and it may even begin after this removal; whereas, if it were a product of nerve irritation, it should be cut short by the removal of its source. Moreover, nerve irritation does produce characteristic and distinct disturbance in the fellow eye, and this disturbance is immediately cut short by the removal of the exciting eye, and never begins or recurs after this removal. The inflammatory disease cannot be regarded as a further development of the neurosis in question, for although the two conditions may occur together, either may occur in maximum intensity entirely apart from the other.

The bacterial theory, on the other hand, explains all these characteristics without difficulty. It is not refuted by the fact that neither optic neurotomy nor neurectomy is a certain safeguard against the disease, for it is proved that the resection-cicatrix is capable of transmitting formed elements. A more cogent objection is the fact that a very large number of eyes which have certainly suffered from microbic infection and inflammation do not induce the disease, and the additional fact that a considerable number of eyes which have induced the disease, have failed under the most careful culture tests to reveal the presence of microbes, although in many cases such experiments have demonstrated their presence not only in the exciting eye, but in the sympathising eye also. The most reasonable explanation of these discrepancies

lies in the fact that we do not know what the particular organism is, or the tests which may be necessary to reveal its presence.

There is little reason for supposing that the ordinary pyogenic coccus is the organism in question. With regard to some other distinct organisms which have been obtained by cultivation from sympathising eyes, it seems yet too soon to decide. A greater number of experiments, and a greater uniformity of results can alone solve the question.

The path along which the germs travel, from the one eye to the other, also remains uncertain. It has been suggested that they enter the general circulation and travel through all parts of the body, but find a suitable home only in the fellow eye. The objection raised against this hypothesis is that it is hardly reconcilable with the long interval between the infection of the first eye and that of the second, and with the infection of the second even after the removal of the first. Another objection is the difficulty of believing that the germs could confine their destructive operation to the eyes. (Neither objection appears to us to be very strong. Have we not in syphilis always a considerable interval between the primary infection and the remote operations of the ubiquitous poison, and have we not in tetanus an example of strict selection of tissue on the part of the poison? A more valid objection seems to be that it is only after infection through the eye that invasion of the other eye occurs. If the germs are carried to the second eye by the general circulation, why should they not be introduced through a wound in any part of the body?—*Rev.*) A direct migration from the one eye to the other appears far more probable. Whether the path lies along the optic nerves or through some other channel, or through both, remains uncertain. The optic nerve route, as already pointed out, is not excluded by the fact that resection of the nerve gives no certain safety to the second eye. Neither is it excluded by the fact that sympathetic inflammation appears commonly to begin as an iritis or

irido-cyclitis, and not as a papillitis, for it is quite probable that in the second orbit the microbes leave the optic nerve and pass forward in the episcleral tissues to enter the eye in the ciliary region. Injection experiments appear to show that the lymph stream, from the brain to the eye, escapes from the nerve in part around the central vessels. Lastly, it is not excluded by the absence of brain infection, for it is not unreasonable to suppose that if the germs reach the intermeningeal spaces they find here an unsuitable soil. It would seem impossible to determine the route with absolute certainty until we can recognise the germs with the microscope, and have the opportunity of completely examining the contents of both orbits, and of the cranial cavity in a recent case of sympathetic inflammation.

Conclusions concerning Treatment.

An eye which is blind, and which is in a condition to excite sympathetic inflammation, should be excised without waiting for any sign of sympathetic irritation. The conditions under which an eye is apt to cause sympathetic inflammation have been fully discussed in the foregoing sections. If the patient declines excision, as large a piece of the optic nerve as possible should be removed.

No definite rule can be laid down for those cases in which an eye threatens danger to its fellow while it retains some vision itself. The aim must be to estimate the probability of permanent retention of useful sight in the first eye, and the amount of danger to the other, but these are questions to which no amount of care and experience can give a certain answer.

If sympathetic inflammation has already broken out, the exciting eye, if blind, should be removed. The removal of the exciting eye has never been proved to have an ill effect on the sympathetic inflammation, but has on the other hand appeared in many cases to act beneficially in this respect, although the improvement may not be permanent. It is rational to remove the source of

the infection, and thereby to obviate the chance of a further migration of germs from the one eye to the other; moreover, the enucleation cuts short any sympathetic irritation which may be present, and thereby, in all probability, favours the subsidence of the inflammation.

If the sympathetic inflammation breaks out while the exciting eye still retains some vision, the exciting eye must not be excised, for it sometimes happens that the second eye is entirely lost, while the exciting eye recovers more or less, and permanently remains the seeing eye. It may even happen that both eyes may recover useful vision.

The sympathetic inflammation itself appears, according to clinical records, to be best influenced by sweating, mercurial inunction, warm compresses, darkness, atropine, and absolute rest of the eye. Retention of the patient in hospital for several months is the best safeguard against relapse. A sufficiently watchful care can seldom be ensured in the patient's own home. Operative interference must be postponed as long as possible, for any operation during the inflammatory period is followed by increased exudation, and even when the inflammation appears to have completely run its course an operation may light it up again.

The remaining section of Schirmer's work describes his own microscopic studies of the pathological anatomy of the disease. Their substance has been already given in the foregoing sections. It has been impossible in this review to cite the opinions and records of the many writers to whom Schirmer refers, but this is amply done in the original paper. A bibliography, comprising nearly three hundred articles—itself a very valuable contribution to the literature of the subject—completes the work.

P. S.

UHTHOFF (Marburg). On the Anatomical Changes underlying the Ocular Disturbances of Cerebral Syphilis. *Bericht über die 22te Versammlung der Ophthalmologischen Gesellschaft. Heidelberg*, 1892.

This paper is based upon one hundred cases of cerebral syphilis observed by Uhthoff during a period of nine years, in some of the Berlin hospitals and cliniques. Seventeen of the cases terminated fatally, and *post-mortem* examinations were made. A few of this number, which were of unusual neuropathological interest, have already been published by Siemerling and Oppenheim, but are included by Uhthoff in his list.

At the meeting in Heidelberg, Uhthoff's communication consisted chiefly of a description and demonstration of the specimens obtained from the fatal cases. The following paragraphs contain a translation of the abstract published in the report of the Congress; the paper will appear in full in a future number of *Graefe's Archives*.

In two cases there were intra-ocular changes; in one an irido-chorioiditis, with very noticeable vascular changes (peri-vasculitis) in the retina; in the other a gummatous formation in the iris. The optic papillæ, nerves, chiasma and tracts were involved in fourteen of the seventeen cases; in several the anatomical changes found *post-mortem* had not given rise to any disturbance of function. "Choked discs" were present in three cases (once unilateral), optic neuritis in two, simple atrophy of the papillæ in three; in six cases no ophthalmoscopic changes were discovered.

In the orbital portion of the optic nerve were found (*a*) perineural changes varying from simple hydrops of the sheath to an intense descending perineuritis, and interstitial neuritis with secondary atrophy; (*b*) simple descending atrophy. In other instances the nerves appeared normal, although there had been well marked basal affection of the optic apparatus, and in one case obliteration of the central retinal artery. The intracranial portion of the

optic nerves showed (*a*) pronounced gummatous degeneration; (*b*) severe perineuritis, (*c*) slight perineuritic changes.

The chiasma was affected in a comparatively large number of cases; four instances of bitemporal hemianopsia were met with. In one case one optic tract in its entire length was converted into a gummatous mass.

Of the motor nerves, the third suffered much more frequently than the sixth. In eight of the seventeen cases there was third-nerve affection, twice bilateral. The sixth nerve was paralysed in two cases.

The trigeminal nerve was involved in three cases, in one of which neuro-paralytic keratitis occurred.

The facial nerve suffered in some or all of its branches in ten cases, the auditory in three, and the olfactory in two.

J. B. L.

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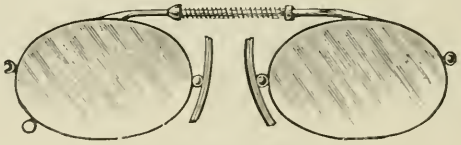

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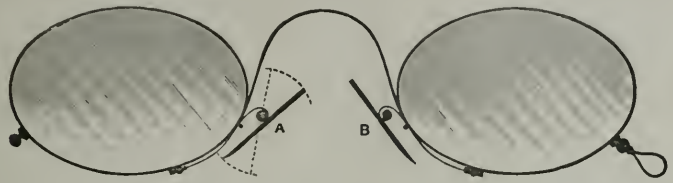

The plaquets A and B are attached to springs, which latter are pushed back by the nose when drawn on. Plaquet A swivels freely as shown by the dotted lines, but plaquet B is fixed at a certain angle and plays an important part. Its inclination must be adjusted to the same angle as THAT side of the wearer's nose. When this is done to suit the wearer, this fixed—and now adjusted—plaquet keeps the Spectalette perfectly horizontal. The free plaquet A adjusts ITSELF to the other side of nose and thus a perfect fit is obtained.

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